NEAR DEATH EXPERIENCES

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The question of a survival after death is one that has fascinated mankind for thousands of years. Although we all try to lead the fullest lives we possibly can, there probably is not an adult person alive who has not wondered what happens when this life ends. Of course, each of the world's major religions offers a unique explanation, and, whether it is the process of reincarnation or the movement into a spiritual realm such as heaven or hell, or even the following of a passing comet, each has its believers. That so many people believe in a life after death is testimony to the fact that there is a large interest in surviving physical death. This fascination may be linked to the curiosity of the unknown, which we all have. As is the case with drugs, sex, and emotions, the dictionary definition of death simply does not give the subjective human being any insight as to what it is. In contrast to those other phenomena, however, death cannot be experimented with. We can try drugs, have sex, and experience emotions, but death seems a little too permanent to mess around with. With that being said, there have been amazing reports of people who have actually experienced death, and have been brought back to life to tell about it. For the most part, these cases have stayed within the realm of parapsychology, appearing every now and then on the cover of Weekly World News or the National Enquirer, next to the typical apocalyptic prophecies and giant baby stories. However, considering the possibilities involved with these cases, should they turn out to be true, combined with the general human interest in the experience of death, it was only a matter of time before science became involved in the investigation of these near-death events. There are currently a number of proposed hypotheses to explain the phenomena of near-death experiences, based on principles of neurochemistry and neurophysiology. The scientific explanations of these phenomena are built upon the implicit assumption that events termed 'death' by the person who experienced them are merely altered states of consciousness. As can be imagined, there are a number of other views about near-death experiences, including the belief that those who had them did actually glimpse into a life after death. While the focus of this discussion will be upon current scientific evidence surrounding near-death phenomenon, the factual information will be considered within the philosophical and theological framework of near-death phenomena, as to avoid a reductionist examination of a very complex occurrence.

Because death is beyond our conscious experience, we dwell on it, and we imagine countless scenarios that may greet us upon dying. The Bible is a source of various descriptions of what

happens after death, many of which shed a positive light on the event:

Romans 8:23 We too, wait anxiously for that day when God will give us...the new bodies he has promised us - bodies that will never be sick again.

2 Corinthians 5:1-3 When we die and leave these bodies, we will have wonderful new bodies in heaven, homes that will be ours forevermore, made for us by God himself.

Central to these accounts is the idea that there is a consciousness, or a soul, that transcends existence of the body on earth. This idea is not limited to the Bible. Plato, the Greek philosopher born in 428 B.C., told a story of a man named Er, whose soul left his body after he died in battle, only to return again after twelve days to revive his body. After coming back to life, Er told stories about what he saw when he was dead:

There were two adjacent openings in the earth, and opposite and above them two others in the heavens, and between them the judges sat. These, having rendered their judgement, ordered the just to go upwards into the heavens...and the unjust to travel downward...with the signs of all their deeds on their backs (Grube 1992).

This passage illustrates the belief that a system of reward and punishment awaits the dead, which is quite a common view, originating perhaps as a "moral enforcer" of behavior - putting forth the belief that good things will come to good people. Again, the story is based upon the idea that the soul is separate from the body, and the soul goes on after the physical death of the body.

Perhaps the most famous writing concerning the mind-body divide was Descartes' Meditations on First Philosophy, in which he called into doubt the existence of his body, positing that he could only know that his mind existed with the famous line, "I think, therefore I am." He then proceeded to 'build up' the world around him, using his sensory observations to confirm that the physical world existed. This method of logic, starting with observations and forming conclusions based on them is called inductive reasoning, and is the basis of modern scientific method (Greyson 1989). There is a problem with trying to use this method to study near-death experiences, however. When people are pronounced clinically dead, they have basically gone beyond the realm of their physical bodies. They are, if anything at all, essentially minds, or souls. At this point, science can only deal with physical bodies, and has no power to confirm the existence of a mind, or a soul, separate from the body. That is, the mind cannot be observed. Modern research deals with this problem by taking the viewpoint that the experience felt by the subjects as 'death' was actually an altered state of consciousness felt while they were alive. The term, near-death experience, then, refers to the experience of this altered state of consciousness.

The introduction of the near-death experience (NDE) to the scientific community can be dated back to 1975, when Dr. Raymond Moody published a book titled Life After Life. Written about actual patients who were pronounced dead and later resuscitated or who came very close to physical death, the book outlines typical characteristics of NDEs. While the book had its greatest effect within popular culture, sparking a wave of interest in mystical experiences and the idea of an afterlife, it did succeed in providing a stimulus for other researchers to look in more depth at the NDEs. In fact, as of 1997, researchers had collected information on over 13 million NDE cases, and the number is continually growing (Morrisey 1997).

Because the NDE is a subjective phenomenon, it has been difficult for researchers to agree upon a set of 'symptoms', or criteria, by which it can be defined. There are always cases in which a NDE did not include one or another of the proposed criteria, which prevent a formal definition from being agreed upon. However, all research has included common characteristics according to which, when measured up against them, a person can be judged as having a NDE. The sensation of being outside of one's physical body is the most commonly reported element of NDEs. Patients either feel that they are observing their own body from above, or that they are leaving their body. Other aspects include: 1) an inexpressibly strong, positive emotional feeling, 2) a slowing of time, 3) a review of the events and people of one's life, and 4) an experience of being in a dark tunnel, at the end of which is a bright light (Moody 1975; Morse et al. 1989; Saavedra-Aguilar and Gomez-Jeria 1989; Greyson 1983; Owens et al. 1990; Jansen 1995). The following description given by a young man who survived a near-fatal car accident illustrates a few of these subjective characteristics:

I saw...the headlights of the car speeding towards us. I heard this awful sound - the side of the car being crushed in - and there was just an instant during which I seemed to be going through darkness, an enclosed space.... Then I was sort of floating about five feet above the street, about five yards away from the car.... I could see my own body in the wreckage among all those people and could see them trying to get it out (Moody 1975).

Another patient, whose vital signs were undetectable after a head injury, reports the following:

I had the feeling of floating in a dark space. The day was bitterly cold, yet while I was in that blackness all I felt was warmth and the most extreme comfort I have ever experienced.... I remember thinking, "I must be dead" (Moody 1975).

First-hand reports such as these can be found throughout all of the published reports on NDEs, including both scientific and non-scientific articles. There is also much evidence to support the fact that people who survive near-fatal situations and experience NDEs have a

much more positive outlook on life after the experience, which leads to the possible therapeutic potential of NDEs (Rosen 1975; Noyes 1980; Jansen 1995).

That accounts of NDEs are so subjective again raises the issue of whether or not scientific research can play a role in explaining such experiences. Much of the research that is done on NDEs, however, is done with the implicit assumption that all psychological phenomena have a neurochemical basis, and that much can be learned about NDEs by studying what goes on in the brain to produce these subjective feelings. The current direction of NDE research is heavily influenced by pioneering studies done by Wilder Penfield in the 1950's that provided sound physical evidence for the link between brain functions and mental states. Penfield was able to induce psychical hallucinations, memories, and religious visions merely by electrically stimulating areas of the cerebral cortex during neurosurgical procedures. There is an extreme example of one patient exclaiming "Oh God, I am leaving my body" when the surface of his temporal lobe was stimulated (Morse 1989). One of the most important conclusions that came out of his work, as applies to near-death studies, is that when complex hallucinations are induced by stimulation of the temporal cortex, they are perceived as real memories by the patients.

Because of the psychical quality of temporal lobe stimulation, much of the early neurological research on NDEs focused on the similarities between them and temporal lobe epilepsy, a condition in which patients are prone to falling into "dreamy states" involving "vivid memory-like hallucinations sometimes accompanied by deja-vu" (Restak 1995). Specifically, temporal lobe epilepsy refers to the condition of sclerotic cell damage in the hippocampus. The hippocampus is a cortical structure in the middle portion of the temporal lobe and is involved in the limbic system, which controls memory, attention, and emotions, among other things (Purves et al. 1997). The "dreamy states" mentioned above refer to seizures characterized by hallucinations of all sensory modalities, motor phenomena such as lip-smacking or twitching, spontaneous emotions, and experiences such as memory flashbacks, depersonalization, out-of-body feelings, and distortions of time and space (Saavedra-Aguilar and Gomez-Jeria 1989; WWW 1;WWW 2).

The striking similarity between these types of seizures and NDEs led researchers to propose a model for the NDEs based on the pathology of temporal lobe seizures. In 1989, neurologists Juan Saavedra-Aguilar and Juan Gomez-Jeria published their model for NDEs, which focused on the rise of neuropeptide and neurotransmitter levels in the brain in response to stressful situations. Basically, the theory was that a traumatic event causes stress on the brain, leading to the release of neurotransmitters and to a decrease in the oxygen tension in the brain (Saavedra-Aguilar and Gomez-Jeria 1989). Many studies were cited that showed that the decreased oxygen levels and the abnormal neurotransmitter and neuropeptide levels contribute especially to disrupt the normal modulation of hippocampal cells in the limbic system. Thus, a stress on the body, such as cardiac arrest or a traumatic accident, could lead to an abnormal excitation of cells involved in the limbic system. As to which neurotransmitters or neuropeptides were directly involved, the researchers suggested endogenous opioid peptides (namely, leu-enkephalin, met-enkephalin, and beta-endorphin), due to "extensive

evidence that they play a fundamental role in the regulation of the excitability of the hippocampus" (Saavedra-Aguilar and Gomez-Jeria 1989). This evidence is also supported by many studies that have shown that during moderate stress and brain trauma, there is a liberation of endogenous peptides within the brain (Saavedra-Aguilar and Gomez-Jeria 1989).

Building upon the model that links NDEs to temporal lobe epilepsy, Dr. Melvin Morse and others proposed a theory based on serotonin rather than opioid peptides (Morse et al. 1989). It is well known that the hippocampus is involved in many serotonergic circuits within the cortex, and that serotonin plays a large role in mood and emotion. The model proposed by Morse and his colleagues suggest that emotional or physical stress, or even psychoactive drugs, could lead to an activation of serotonergic pathways, which innervate areas of the temporal lobe, including the hippocampus. Support for this hypothesis is given by an experiment that measured responses to pain perception in animals when levels of neurotransmitter were altered. After testing dopamine, norepinephrine, and serotonin, only changes in serotonin were found to affect responses to painful stimuli, illustrating that responses to stress involve serotonergic pathways (Morse et al. 1989). The activation of the temporal lobe by serotonin would result in the same effects as documented by Wilder Penfield (hallucinations, memories, and religious visions) after directly electrically stimulating the area. Therefore, many of the aspects of NDEs would be elicited according to this model.

The two proposed models for NDEs based on the pathology of epileptic seizures realize that a stress on the brain can induce neurochemical changes within the temporal lobe. These changes result in overactivity, which manifests itself as hallucinations and memories for the patient. With temporal lobe seizures, however, the initiating factor is an actual location in the brain where cell death occurs. In the proposed models for NDEs, on the other hand, the initiating factor is a "stress on the brain". This can be the result of any number of traumatic events, and has yet to be clearly defined by anyone in the scientific community. No physical explanation for the flood of chemicals in the brain has been proposed. There are also practical objections that seem to limit the effectiveness of each of the theories' ability to fully explain the aspects associated with NDEs. To begin with, the neuropeptides implicated in the first study are known not to be potent hallucinogens. Also, injection of the neuropeptides into the central nervous system has analgesic (pain-killing) effects which last over twenty hours, while the near-death experience is usually quite rapid (Jansen 1995).

In comparison to the opioid peptide hypothesis, the serotonin model seems quite plausible. Indeed, the objections to it are less sound, but worth considering, nonetheless. A weak argument against the involvement of serotonin as the main cause of NDEs is that the number of serotonergic cells in the cortex is very small in comparison to the number cells that use other neurotransmitters such as GABA, and more importantly, glutamate (Jansen 1995). This line of reasoning does not seem to hold much relevance, considering the intensity of hallucinatory effects that a small amount of a serotonin agonist, such as LSD, can have. On the other hand, the types of hallucinations provided by classical psychedelics (all of which affect serotonergic pathways) are fundamentally different from the typical NDEs. Psychedelic, or "mind-manifesting", serotonin agonists such as LSD and DMT cause an increased

awareness of external sensory perceptions and can result in "synethesia", which is the mixing of sensory information from different pathways. In near-death experiences, however, there is a marked decrease in sensory input from the environment, as evidenced by the frequency of reports of a dark tunnel with a bright light at the end of it. Psychedelics also often cause increased anxiety, while near-death experiences are almost always accompanied by strong feelings of peace and contentment. Furthermore, while LSD has been noted to cause feelings of being at one with the universe, or "ego-loss," there are rarely experiences with psychedelics that come close to the depersonalization and out-of-body experiences that are characteristic of NDEs (Jansen 1995). Thus, while the serotonin model is plausible and may contribute to NDEs, it does not seem to provide sufficient evidence that NDEs are mainly a result of the stimulation of serotonergic pathways to the temporal lobe.

Taking into account the problems with the previous two hypotheses, the most recent model for the neuropathology of NDEs is based upon the similarities between NDEs and ketamine, a 'dissociative' anaesthetic. Marketed by Parke Davis as Ketelar, ketamine is a cheap prescription drug used as a general anaesthetic by veterinarians (WWW 3). Because of its affordability and because it is not a respiratory depressant, as opposed to other common anaesthetics, it is also commonly used during surgical procedures on children in third world countries (Jansen 1995; Grinspoon and Bakalar 1997). The anaesthetic effects are a result of the patient being so 'dissociated', or 'removed from their body' that it is possible to carry out surgical procedures. It is commonly administered with sedatives to produce unconsciousness in the patient, avoiding any harmful psychological reaction to the dissociative state (Jansen 1995). For surgeries, the common dosage is approximately 10 milligrams per kilogram of body weight, administered intramuscularly. However, when taken at 1/10 that amount (sub-anaesthetic doses), ketamine can provide for a very deep hallucinatory state that, in many respects, resembles the near-death experience. In other words, a 50-100mg intramuscular dose of ketamine can cause feelings of being disconnected from the environment, feelings of floating and disembodiment, the experience of traveling through a dark tunnel, and a reliving of earlier life events (Grinspoon and Bakalar 1997).

An examination of first-hand accounts of experiences with ketamine can provide a good indication of the similarity between the subjective experience on the drug and the NDE.

I was convinced I was dead. I was floating above my body. I reviewed all of the events of my life and saw a lot of areas where I could have done better (Jansen 1995)

A patient who was given ketamine experimentally as a therapeutic intervention for his alcoholism said the following:

I found myself inside a gigantic tunnel whose mouth reached a terrifying height.... it was me who was rushing towards nothing. But at the same time, I regarded myself as a detached spirit, as if I were split apart (Krupitsky and Grinenko 1997).

Another patient in the same study commented:

Several scenes of my life passed before my eyes. They were from my childhood and youth, everything in reddish-brown colors.

Another example contrasts the ketamine experience to that of LSD:

...becoming a disembodied mind or soul, dying and going to another world. Childhood events may also be relived. The loss of contact with ordinary reality and the sense of participation in another reality are more pronounced and less easily resisted than is usually the case with LSD. The dissociative experiences often seem so genuine that users are not sure they have not actually left their bodies (Jansen 1995).

Timothy Leary, one of the 1960's psychedelic pioneers, called his experiences with ketamine "experiments in voluntary death" (Leary 1983). All studies done on the effects of ketamine seem to agree that the dissociative experience provided by this drug are of a vastly different quality than the perceptual distortions provided by LSD. Moreover, in examining the above accounts, it becomes strikingly clear that users of sub-anaesthetic doses of ketamine experience some of the same events as patients who survive NDEs.

The recently discovered mechanism of ketamine action on the brain provides both an account of the differences between ketamine and LSD and a model for the main neurochemical mechanisms of NDEs. The main excitatory neurotransmitter in the cerebral cortex is glutamate. Glutamate acts by binding post-synaptically to NMDA receptors, which are multisubunit proteins that form ion channels selective for calcium and sodium. When glutamate binds, the channel can open and allow the cations to flow in and contribute to the depolarization of the cell. However, if glutamate is present in excessive amounts, the ion channel may stay open too long, causing cations and, subsequently, water, to rush in and cause the cell to burst. This process of cell death is called excito-toxicity, and can occur in conditions of low oxygen, low blood sugar, and epilepsy. Ketamine can prevent this process of cell death from occurring. It binds to a site of the NMDA receptor called the 'PCP' site, which is located inside the ion channel. This is a different site than the glutamate binding site, and ketamine is thus a non-competitive antagonist of NMDA receptors. Even if glutamate is present in excess amounts, the presence of ketamine will result in the blockage of the ion channel and the prevention of excitotoxicity. It is also this mechanism - the binding of

ketamine to the 'PCP' site of NMDA receptors in the cerebral cortex - that is responsible for the drastic hallucinations that follow intramuscular administration of sub-anaesthetic doses of the drug (Jansen 1995).

Since the glutamate receptors are more widespread and more prevalent than serotonin receptors within the cortex, it can be imagined that blockage of these receptors would cause more drastic effects on consciousness than stimulation of serotonin receptors, which could explain the difference between ketamine-induced hallucinations and LSD-induced distortions. Of course, the mechanism of ketamine itself is not sufficient to explain near-death experiences, since ketamine is not usually present in patients who experience near-death phenomena. However, the effects of ketamine, as learned from accounts of users, compare quite similarly to the qualities of NDEs, and thus lead to the speculation of a common mechanism. In fact, researchers now believe there may be some endogenous substance(s) within the brain that may block NMDA receptor ion channels at the 'PCP' site in the same manner that ketamine does. In 1995, psychiatrist Karl Jansen proposed that such substances could be the result of the "evolutionary development of protective mechanisms against excitotoxicity". That is, the chemicals may have conferred an advantage in people, protecting them against cell death during times of excess glutamatergic activity in the cortex. He also proposes that some individuals may have a more advanced protective mechanism, which would explain the subjective differences in NDEs, and the occurrence of NDEs in some patients who were really not even in danger of dying. Evidence of substances, called "endopsychosins," that bind to the PCP site and are found in the brain has been found (Jansen 1995, 1997). Also, ions such as magnesium and zinc bind within the ion channel of NMDA receptors (although not at the PCP site), blocking the inflow of ions even when the channel is open. Either the "endopsychosins" or the large ions could be responsible for the main qualities of NDEs.

Although the main connection between the ketamine model is speculative, since neither endopsychosins nor magnesium or zinc has been proven to recreate NDEs, it does seem to provide a solid neurophysiological model for the phenomena. One criticism of the model, which actually could apply to all three of the models mentioned thus far, is that recreating the features of an experience is not at all equivalent to recreating the experience itself. To put it another way, not every sweet, shiny, round object from Washington is an apple. In each hypothesis, it has been assumed that reducing the NDE to a collection of its component parts and then simulating those component parts is the same as simulating the NDE. That is simply not the case with any subjective phenomenon. An experiment that could be done in the future is to administer sub-anaesthetic doses of ketamine to people who have already had an NDE and determine whether the subjective feel of the experiences is the same. Even this, however, would leave room for objections since the experiences and the viewpoints of people constantly change. Things that they would consider pretty and exciting one day may be ugly and dull a week later. Thus, the subjective feel of an experience is open to interpretation even within the person experiencing it.

This brings the discussion back to the limits of science in understanding NDEs. All the research on NDEs ignores the question that the majority of non-scientists are most interested in.

Namely, is there life after death? Rather than focus on that aspect of NDEs, scientists are quick to dismiss the possibility that the events the NDE subject recalls occurred during the time he/she was dead. Instead, it is assumed that the events - the dark tunnel, the bright light, the peaceful feelings, the life-recall - all occurred while the patient was alive, in an altered state of consciousness. However, it should not be expected of scientific research to examine such issues as life after death. As stated, science is based on knowledge that can be gained by observation. Perhaps in pursuing questions like what causes NDEs, science can evolve into something greater, and eventually gain the tools for looking into the unobservable, mystical aspects of life, but for now it is limited by its methods. Those who believe that the NDE was a real glimpse into the afterlife are skeptical that science will ever be able to reduce the experience down to molecules and pathways. Researchers, on the other hand, feel a reverse-skepticism towards the possibility that subjects can have a conscious experience while physically dead. Until these two views can be reconciled, and a greater understanding of the NDE is worked towards from both directions, it seems little progress can be made in understanding the nature and the implications of the NDE.

References

Greyson, Bruce. MD. The Psychodynamics of Near-Death Experiences. The Journal of Nervous and Mental Disease: Vol. 171 No.6, 376-381 (1983).

Greyson, Bruce, MD. Editorial: Can Science Explain the Near Death Experience? Journal of Near Death Studies: Vol.8 No.2, 77-92 (Winter 1989).

Grinspoon, Lester and James B. Bakalar. Psychedelic Drugs Reconsidered. New York: the Lindesmith Center (1997).

Grube, G.M.A, trans. Plato: the Republic. Indianapolis: Hackett Publishing Company, Inc. (1992).

Jansen, Karl L.R. Using Ketamine to induce the near-death experience: Mechanism of Action and Therapeutic Potential. Yearbook for Ethnomedicine and the Study of Consiousness: Issue 4, 55-81 (1995).

Jansen, Karl L.R. MD, PhD, MRCPsych. A Ketamine Model of the Near Death Experience. Journal of Near-Death Studies: Vol. 16 No.1 (Fall 1997).

Krupitsky, E.M. MD. and A.Y. Grinenko, MD. PhD. Ketamine Psychedelic Therapy (KPT): A Review of the Results of Ten Years of Research. Journal of Psychoactive Drugs: Vol. 29 No.2, 165-183 (April-June 1997).

Leary, Timothy F. Flashbacks: An Autobiography. Los Angeles: J.P. Tarcher (1983).

Moody, Raymond A., Jr. MD. Life After Life. New York: Bantam Books (1975).

Morrisey, Dianne, PhD. You Can See the Light. New Hampshire: Stillpoint Publishing (1997).

Noyes, Russell, Jr. Attitude Change Following Near-Death Experiences. Psychiatry: Vol. 43, 234-252 (August 1980).

Owens, J.E., et al. Features of "near-death experience" in relation to whether or not patients were near death. Lancet: Vol. 336, 1175-1177 (1990).

Purves, Dale, et al., ed. Emotions. Chapter 27 (pp 513-527) in Neuroscience. Massachusetts: Sinauer Associates, Inc. (1997).

Restak, Richard MD. Complex Partial Seizures Present Diagnostic Challenge. Psychiatric Times: Vol. XII Issue 9, (1995).

Rosen, David H. MD. Suicide Survivors. The Western Journal of Medicine: Vol. 122 No.4, 289-294 (April 1975).

Saavedra-Aguilar, Jaun C. MD. and Juan S. Gomez-Jeria, Lic.Q. A Neurobiological Model for Near-Death Experiences. Journal of Near-Death Studies: 7(4), 205-222 (Summer 1989).

WWW 1. Temporal lobe seizure disorder response in the Neurology and Neurosurgery forum.

WWW 2. American Academy of Neurology. Temporal lobe epilepsy is characterized by seizures causing a disturbance of brain activity.

WWW 3. Ketamine Hydrochloride FAQ Page.

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